159 PILOT STUDY EXPLORING THE REGIONAL REPOLARISATION INSTABILITY INDEX IN RELATION TO MYOCARDIAL HETEROGENEITY AND PREDICTION OF VENTRICULAR ARRHYTHMIA AND DEATH

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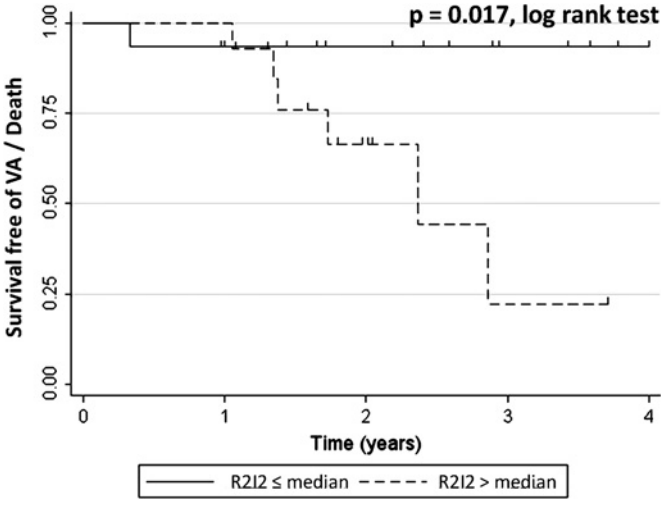
Introduction There is a need for better sudden cardiac death (SCD) risk markers. Mounting evidence suggests that the mechanism underlying risk of ventricular arrhythmia (VA) is increased heterogeneity of electrical restitution. We investigated a novel measure of action potential duration (APD) restitution heterogeneity: the Regional Repolarisation Instability Index (R2I2) and correlated it with peri-infarct zone (PIZ) a cardiac magnetic resonance (CMR) anatomic marker of VA risk.

Methods Blinded retrospective study of 30 patients with ischaemic cardiomyopathy assessed for an implantable cardioverter defibrillator. The R2I2 was derived from high resolution 12 lead ECG recorded during programmed electrical stimulation (PES). ECG surrogates were used to plot APD as a function of diastolic interval; the R2I2 was the maximal value of the mean squared residuals of the mean points for anterior, inferior and lateral leads normalised to the mean value for the total population. PIZ was measured from late gadolinium enhanced CMR images using the full width half maximum technique.

Results Seven patients reached the endpoint of VA/death (median follow-up 24 months). R2I2 > median was found to be predictive of VA/death independent of PES result, left ventricular ejection fraction and QRS duration (6/14 vs 1/15 p=0.031). Modest correlation was seen between the R2I2 and PIZ (r=0.41 p=0.057) (Abstract 159 figure 1).



Abstract 159 Figure 1

Conclusions In this pilot study of ischaemic cardiomyopathy patients, the R2I2 was shown to be an electrical measure of VA/death risk with a moderately strong correlation with an anatomic measure of arrhythmic substrate, the extent of PIZ. The R2I2 may add value to existing markers of VA/death and merits further investigation.

Abstract 159 Table 1

Variable	Whole Group (n=30)	No VA/death (n=23)	VA/death (n=7)	p
Age (years)	67±9	65±9	72±8	0.055
Sex (% male)	97	96	100	
QRSD(ms)	107±20	107±21	106±15	0.95
EF(%)	31±14	32.4±15	27±7.5	0.34
PES result (positive/total)	12/30	7/23	5/7	0.068
R2I2>median	14/29	8/22	6/7	0.031
EDV index (ml/cm)	1.48±0.41	1.49±0.41	1.45±0.45	0.84
SV index (ml/cm)	0.42±0.14	0.43±0.14	0.39±0.15	0.47
Follow-up (months)	24 (18)	24 (16)	16 (16)	0.088
PIZ %	7.8 (10.7)	7.5 (8.4)	13.6 (8.5)	0.093
Scar %	10.9 (16.5)	9.67 (13.5)	21.9 (17.8)	0.16

160 HIGH DOSE OCTREOTIDE; A NOVEL THERAPY FOR THE TREATMENT OF DRUG REFRACTORY POSTURAL ORTHOSTATIC TACHYCARDIA SYNDROME IN PATIENTS WITH JOINT HYPERMOBILITY SYNDROME

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Introduction Postural orthostatic tachycardia syndrome (POTS) is defined as symptomatic orthostatic intolerance with an increase in heart rate of 30 beats per minute within 10 min of head up tilt (HUT). This dysautonomia causes wide-ranging symptoms including palpitations, presyncope, chronic fatigue, headache and cognitive difficulties. When POTS occurs in patients with pre-existing Joint Hypermobility Syndrome (JHS), symptoms begin approximately a decade earlier than non-JHS patients with a preponderance of neurological features, secondary to cerebral hypoperfusion. Vascular laxity with splanchnic venous pooling has been implicated as a causative factor thus measures to expand plasma volume (thereby increasing mean arterial pressure and restoring cerebral perfusion) form the mainstay of therapy. Symptomatic improvements have been previously reported in POTS patients with the somatostatin analogue Octreotide, a powerful splanchnic vasoconstrictor. We report the first UK series of JHS patients with drug refractory POTS treated with high-dose octreotide.

Methods Six patients (female, aged 21–52) were referred to our institution. All had known JHS (4 requiring a wheelchair), neurological symptoms (headache and cognitive impairment) and diagnostic tilt-table testing with a mean increase in heart rate of 64 beats/min (range 47–73) with head-up tilt (HUT). All patients had remained symptomatic despite pre-treatment with a mean of 5 POTS medications (range 5–7) including fludrocortisone, midodrine, propranolol, ivabradine, selective serotonin reuptake inhibitors, gabapentin and erythropoietin. Octreotide was commenced using a short-acting preparation given 3 times daily (dosage 50–250 μg according to body mass) in conjunction with a long-acting (monthly), intramuscular injection (dosage 10–30 mg). The short-acting preparation was weaned following the second monthly injection.

Results During follow-up of 3 months (range 1–8), 3 (50%) patients reported a complete resolution of all postural and neurological symptoms which corresponded with a normalised response to HUT. The remaining patients reported a dramatic improvement but ongoing postural symptoms. No patients developed supine hypertension. Side effects including mild abdominal discomfort and transient diarrhoea were reported in 3 (50%) patients.

Conclusion Octreotide is increasingly recognised as an effective therapy in POTS patients. Both short-acting, subcutaneous (0.9 μg/Kg) and long-acting, intramuscular (10–20 mg) preparations have

previously been reported. We conclude that higher dosages of both preparations when administered together are effective and well tolerated in JHS patients with drug refractory POTS.

161 CATHETER ABLATION OF ATRIAL FIBRILLATION ON UNINTERRUPTED WARFARIN USING STANDARD AND DUTY CYCLED RADIOFREQUENCY ENERGY: SAFE AND EFFECTIVE

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Introduction Catheter ablation (CA) for atrial fibrillation (AF) is growing exponentially. Although ablation for paroxysmal AF (PAF) is associated with shorter procedure times and less extensive left atrial ablation vs persistent AF thromboembolic complications can occur in both sub-groups. Inadequate anticoagulation leads to thrombotic complications and excessive anticoagulation can lead to bleeding risks. Many centres adopt a policy of discontinuing warfarin in the immediate run-up to the procedure, covering the procedure with unfractionated heparin and "bridging" postoperative patients with low molecular weight heparins (LMWH) back onto warfarin. We wished to determine the safety of CA for AF with a therapeutic INR using both the single transseptal approach and duty cycled radiofrequency energy (RF) with non irrigated PVAC catheters and the double transseptal puncture technique using irrigated RF catheters and either CARTO or NAVX electroanatomical mapping.

Methods A retrospective analysis of 173 patients who underwent CA for AF while taking uninterrupted warfarin. Procedural target International Normalised Ratio (INR) was 2–3 with a peri-procedural target ACT of 300–350 s. In sub therapeutic INR patients weight adjusted LMWH was used post procedure with warfarin until INR was >2. Standard technique employed was large area circumferential ablation using conventional RF energy or pulmonary vein isolation using duty cycled RF energy. Data was gathered for demographics, procedural INR, total dose of unfractionated heparin, fluoroscopy time, and type of radiofrequency energy used. Endpoints were minor bleeding, major bleeding (requiring transfusion), vascular complications, pericardial tamponade and stroke/TIA within 28 days of the procedure.

Results There were 128/173 male patients, age range between 21 and 73 years (mean 57 years). 122 underwent ablation for PAF and 51 for persistent AF. Mean procedural INR was 2.4 (range 1.7–3.9). Mean unfractionated heparin dose was 6000 units (range 1000–14 500). Mean fluoroscopy time for the PVAC group was 23.4 mins (range 8.3–50.1 mins). Mean fluoroscopy time for CARTO/NAVX group was 31mins (range 14.10–58.44 mins). There were no major bleeding complications. There was 1 minor bleeding complication with a groin pseudoaneurysm. There were 2 cases of pericardial tamponade (2/173%–1.2%) both managed with percutaneous pericardial drainage. There were no stroke/TIAs.

Conclusion These data demonstrate that CA for AF by both single and double transseptal technique using both standard RF and duty cycled RF while maintaining a therapeutic INR is a safe procedure. Maintaining a therapeutic INR reduces the risk of embolic events associated with "bridging" heparin without an increase in bleeding complications. This technique is convenient for patients and avoids switching between LMWH and warfarin and ensures patient safety by maintaining therapeutic anticoagulation before, during and after the procedure.

162 THE CLINICAL MANAGEMENT OF RELATIVES OF YOUNG SUDDEN ARRHYTHMIC DEATH VICTIMS; ICDS ARE RARELY INDICATED

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Introduction Following National Service Framework guidance on the management of sudden cardiac death (SCD), regional inherited cardiac conditions clinics were established to identify and treat those at increased risk of dying from an arrhythmic condition. Studies have examined the yield of different diagnostic strategies but the outcome of management in these patients has not been reported.

Methods We present data on 193 consecutive patients (108 families) referred to a regional inherited cardiac conditions clinic because of SCD/aborted cardiac arrest of a relative. The mean age on referral was 38±17 yrs and mean duration of follow-up was 15 months (range 1 day to 56 months). All individuals underwent clinical assessment by history, examination, ECG and echo. If treadmill exercise test had not previously been performed this was undertaken. Further imaging by CMR or contrast echo was performed in those with structurally abnormal hearts or with T wave inversion in V_2/V_3. Ajmaline provocative testing was performed in those with a history and/or ECG suggestive of Brugada syndrome.

Results Of the 193 patients, 43 individuals (22%) from 36 families (33%) were diagnosed with an inheritable cause of SCD and 145 patients were clinically normal (see Abstract 162 table 1). Five patients were found to have other conditions (LV non-compaction, AVNRT, skeletal myopathy, mild AS and congenital sub-aortic membrane). Of the 43 patients diagnosed with an inheritable condition, 21 had medication commenced by the clinic (β-blockers (21), ACEi/ARB(2), Spironolactone[1]). ICDs were implanted as per HRUK guidelines, resulting in 4 patients having an ICD inserted on clinic recommendation (2 HCM, 1 DCM, 1 ARVC). To date no appropriate therapies have been administered (follow-up 8–29 months) but there was 1 inappropriate shock from a fractured lead. Three individuals had β-blockade withdrawn after negative genetic testing for an established familial mutations (2 CPVT, 1 LQT), one ICD was removed and one deactivated (both negative for CPVT). Of the 145 patients thought to be clinically normal, 85 were reassured and discharged, 13 failed to return to clinic and 47 are regular reviewed as the risk of developing an inheritable condition cannot be excluded; this includes those with family histories of HCM (7), ARVC (12), DCM (9), CPVT (5), Brugada (4) and LQT(1). To date no deaths have occurred in those diagnosed with inherited causes of SCD (follow-up mean 20, 1–52 range) or those clinically normal with ongoing review (follow-up mean 22 months, 1–56 range).

Abstract 162 Table 1

Diagnosis of patient	Numbers
Clinically normal	145
LQTS	12
Brugada	2
CPVT	5
ARVC	7
DCM	7
HCM	10

Conclusion A diagnosis of an inheritable cause of SCD was obtained in 22% of individuals and 33% families with a history of SCD/aborted cardiac arrest in a relative. The number of ICDs inserted was very small (2%) and there have been no appropriate therapies in this